Supporting Information

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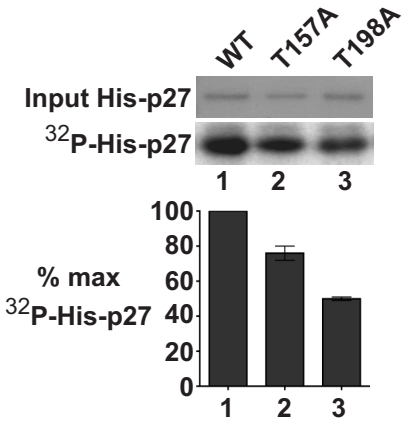

Fig. S1. Recombinant WT and mutant His-p27 proteins were reacted with RSK1 kinase in the presence of [γ-^{32}P]ATP. Coomassie stain of the input p27 and radioactivity in His-p27 bands are shown. Kinase activity (mean of 4 experiments $\pm$ SEM) is graphed as % maximum (% max), using radioactivity in His-p27WT as 100% maximum kinase activity.

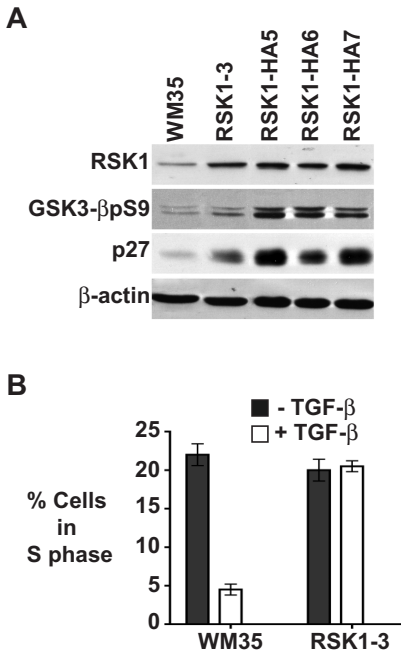

Fig. S2. RSK1 overexpression confers TGF-β resistance. WM35 cells were transfected with WT-RSK1 or HA-RSK1. (*A*) RSK1-transfectants show increased RSK1, GSK3-β phosphorylation (GSK3-βpS9), and increased p27. (*B*) Flow cytometric analysis of WM35 and RSK1-3 clone treated with (□) or without (■) TGF-β for 48 h. Graph shows % cells in S phase ± SEM.

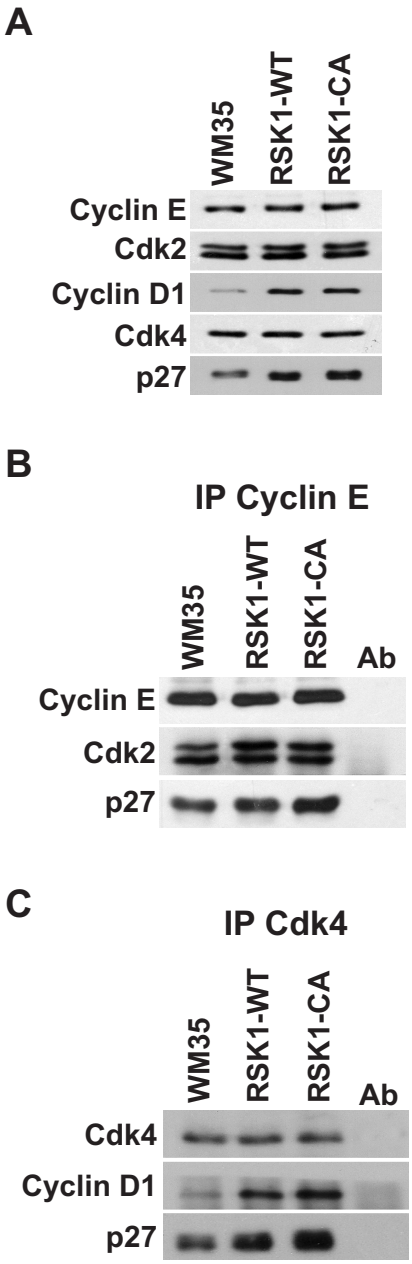

Fig. S3. RSK1 increases p27-cyclin D-Cdk4 complexes. WM35 cells were infected with pBabe RSK1-WT or RSK1-CA. (*A*) Levels of cyclin E, Cdk2, cyclin D1, Cdk4, and p27 in proliferating cells were analyzed by Western blotting. (*B* and *C*) Cyclin E (*B*) and Cdk4 (*C*) immunoprecipitates were resolved and associated proteins were analyzed by blotting. Ab indicates antibody-only control.